Sleep Medicine 15 (2014) 1586–1589

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Brief Communication

# Delayed sleep timing is associated with low levels of free-living physical activity in normal sleeping adults

Ari Shechter, Marie-Pierre St-Onge *

*New York Obesity Research Center, Columbia University, New York, NY, USA*

ARTICLE INFO

*Article history:*
Received 21 May 2014
Received in revised form 24 June 2014
Accepted 14 July 2014
Available online 2 September 2014

*Keywords:*
Sleep timing
Chronotype
Sleep quality
Accelerometry
Physical activity
Obesity

ABSTRACT

*Objective and Background:* We and others have reported that experimentally induced short sleep does not affect resting metabolic rate and leads to increased laboratory-measured 24-h energy expenditure. Here, we aimed to determine if sleep timing and/or quality are related to physical activity (PA) levels.
*Methods:* Measures of PA via waist actigraphy, sleep diary, and sleep quality questionnaires were collected over a 7–18-day period in 22 adults (mean age ± standard deviation (SD): 35.8 ± 4.6 years, and mean body mass index ± SD: 23.8 ± 1.1 kg/m$^2$) who were on their habitual sleep–wake and activity schedules.
*Results:* During the recording period, mean (±SD) bedtime and wake times were 00:17 ± 1:07 h (range: 22:02–02:07 h) and 08:20 ± 1:14 h (range: 06:30–10:11 h), respectively. After controlling for sleep duration, later bedtime, wake time, and midpoint of sleep were associated with less time spent in moderate-to-vigorous PA ($p = 0.013$, $p = 0.005$, and $p = 0.007$, respectively), and increased time in sedentary PA ($p = 0.016$, $p = 0.013$, and $p = 0.013$, respectively).
*Conclusions:* Current results suggest that even relatively small alterations in sleep timing may influence PA. However, causality cannot be inferred from this cross-sectional study. Clinical intervention studies should be conducted to assess the relationship between sleep timing and energy balance.


## 1. Introduction

In attempting to explain the link between short sleep and obesity [1], we have demonstrated that experimental sleep restriction affects appetite-regulating hormones [2], increases energy intake [3], and increases in-laboratory-measured 24-h energy expenditure [4]. However, relatively little work has been done to determine how habitual sleep patterns influence physical activity (PA) levels. This is relevant as sleep–wake behavior, particularly sleep timing, is associated with food choice [5] and energy intake [6] in children/adolescents. Late bedtime and wake time are associated with lower moderate-to-vigorous PA (MVPA) levels compared to earlier sleep timing in children/adolescents [7]. The timing of the sleep episode may be an important determinant of energy balance [8]. Therefore, we aimed to determine the relationship between sleep timing and quality and PA levels, under habitual conditions in adults. We hypothesized that later sleep timing, worsened sleep quality, and greater daytime somnolence would be associated with less vigorous and more sedentary PA levels.

## 2. Methods

This is a retrospective analysis of baseline screening data from a previous experiment on the effects of sleep restriction on energy balance [3,9]. Participants were 30–45 years of age, with a body mass index (BMI) between 22 and 26 kg/m$^2$. Exclusion criteria included smoking, type 2 diabetes, history of alcohol or drug abuse, caffeine intake >300 mg/d, shift work, trans-meridian travel, and any eating, sleeping, or neurological disorders.

As part of the pre-laboratory screening procedures [3], participants had their sleep and PA tracked for 7–18 d while they were on their habitual schedules. Actigraphy was recorded with GT3x ActiGraph monitors (Actigraph LLC, Pensacola, FL, USA), a triaxial accelerometer that monitors PA and quantifies energy expenditure in step counts per minute (cpm). Participants continuously wore the actigraph at the waist level by attaching it to an elastic belt. The sampling rate was 60 Hz, and data were reintegrated into 60-s epochs for scoring. Actilife5 software (Actigraph LLC, Pensacola, FL, USA) employing Freedson cut points [10] was used to quantify PA levels. Sedentary was defined as <100 cpm, light activity was 100–1951 cpm, moderate activity was 1952–5725 cpm, and vigorous activity was ≥5725 cpm. Moderate and vigorous were combined to form the category MVPA [11].

*Abbreviations:* ESS, Epworth Sleepiness Scale; MEQ, Morningness–Eveningness Questionnaire; MVPA, moderate-to-vigorous physical activity; PA, physical activity; PSQI, Pittsburgh Sleep Quality Index; TST, total sleep time.

**Clinical Trial Registration:** Trial registration on http://www.clinicaltrials.gov/show/NCT00935402.

* Corresponding author. 622 West 168th Street, PH9E-105Q New York, NY 10032, USA. Tel.: +1 212 305 5960; fax: +1 212 305 8466.
*E-mail address:* ms2554@columbia.edu (M.-P. St-Onge).

http://dx.doi.org/10.1016/j.sleep.2014.07.010
1389-9457/

**Table 1**
Characteristics, sleep, and physical activity data of study participants.

| | Range, $n = 22$ | All, $n = 22$ | Males, $n = 16$ | Females, $n = 6$ | $p$ value |
|---|---|---|---|---|---|
| Age, years | 30–45 | 35.8 ± 4.6 | 36.2 ± 5.0 | 34.7 ± 3.3 | 0.50 |
| BMI, kg/m$^2$ | 22–25 | 23.8 ± 1.1 | 23.9 ± 1.1 | 23.3 ± 1.2 | 0.28 |
| Recording length, days | 7–18 | 12.5 ± 2.9 | 12.8 ± 2.5 | 11.5 ± 4.0 | 0.36 |
| Bedtime, h | 22:02–02:07 | 00:17 ± 1:07 | 00:12 ± 1:13 | 00:30 ± 0:51 | 0.58 |
| Wake time, h | 06:30–10:11 | 08:20 ± 1:14 | 08:15 ± 1:21 | 08:33 ± 0:56 | 0.60 |
| Midpoint of sleep, h | 02:02–06:00 | 04:19 ± 1:09 | 04:13 ± 1:15 | 04:32 ± 0:52 | 0.58 |
| TST, min | 389–556 | 448.8 ± 30.9 | 451.0 ± 36.1 | 442.78 ± 6.71 | 0.59 |
| PSQI score | 0–3 | 1.4 ± 1.1 | 1.4 ± 1.0 | 1.3 ± 1.0 | 0.94 |
| ESS score | 0–11 | 3.3 ± 2.7 | 3.3 ± 2.8 | 3.3 ± 2.5 | 0.95 |
| MEQ score | 39–73 | 56.9 ± 8.2 | 56.6 ± 8.8 | 57.5 ± 7.1 | 0.94 |
| time in sedentary PA, % | 55.8–95.9 | 83.2 ± 8.3 | 83.3 ± 8.8 | 82.8 ± 7.6 | 0.90 |
| time in light PA, % | 3.7–28.7 | 13.6 ± 5.8 | 13.5 ± 5.9 | 14.1 ± 6.1 | 0.84 |
| time in MVPA, % | 0.4–15.5 | 3.2 ± 3.1 | 3.2 ± 3.6 | 3.1 ± 1.8 | 0.96 |

*Abbreviations:* BMI, body mass index; ESS, Epworth Sleepiness Scale; MEQ, Morningness–Eveningness Questionnaire; MVPA, moderate-to-vigorous physical activity; PA, physical activity; PSQI, Pittsburgh Sleep Quality Index; TST, total sleep time.
Data are mean ± SD.

The software was also used to determine total sleep time (TST) [12], with bedtime and wake time determined from sleep–wake logs. The midpoint of the sleep episode was calculated as wake time minus half the total time in bed. Chronotype (i.e., either having a morning or evening preference) was assessed with the Horne–Ostberg Morningness–Eveningness Questionnaire (MEQ) [13], daytime sleepiness with the Epworth Sleepiness Scale (ESS) [14], and sleep quality with the Pittsburgh Sleep Quality Index (PSQI) [15]. Procedures were approved by St. Luke's-Roosevelt Hospital and Columbia University Institutional Review Boards, and participants provided written informed consent.

Multiple regression analysis examined the associations between sleep and PA. Analyses were adjusted for sex, age, BMI, and waist-actigraphy-derived TST. Actigraphy-based estimates of TST have been validated in adults with the use of wrist-bound accelerometers and not waist-mounted actigraphs, as used here. Therefore, we utilized the observed TST metric as a proxy measure of sleep duration, and it was included in the models only as a covariate to control for approximate time spent asleep. Data showed homoscedasticity and there was no multicollinearity among variables. Analyses were performed using Statistical Package for the Social Sciences (SPSS) Version 22 (IBM, Armonk, NY, USA). Data are expressed as mean ± SD. Statistical significance was defined as $p < 0.05$.

## 3. Results

Twenty-two participants were included (Table 1). Bedtime and wake time were 00:17 ± 1:07 h (range: 22:02–02:07 h) and 08:20 ± 1:14 h (range: 06:30–10:11 h), respectively. The midpoint of sleep was 04:19 h ± 1:09 h (range: 02:02–06:00 h). The participants were good sleepers (PSQI: 1.4 ± 1.1, range: 0–3) with minimal daytime sleepiness (ESS: 3.3 ± 2.7, range: 0–11). The MEQ score was 56.9 ± 8.2 (range: 39–73), indicative of an intermediate chronotype. Two participants were definite morning types (scores: 70, 73), six were moderate morning types (range: 61–68), 13 were intermediate types (range: 46–57), and one was a moderate evening type (score: 39). Having a later chronotype, reflected by lower score on the MEQ, was associated with later bedtime ($r = –0.74$, $p < 0.001$), wake time ($r = –0.73$; $p < 0.001$), and midpoint of sleep ($r = –0.75$, $p < 0.001$).

TST was 448.8 ± 30.9 min (range: 389–556 min). The participants spent 83.2 ± 8.3% (range: 55.8–95.9%) of their time in the sedentary state, 13.6 ± 5.8% (range: 3.7–28.7%) in light PA, and 3.2 ± 3.1% (range: 0.4–15.5%) in MVPA. TST was not associated with bedtime ($r = –0.09$, $p = 0.70$), wake time ($r = 0.26$, $p = 0.24$), or midpoint of sleep ($r = 0.10$, $p = 0.66$).

After controlling for age, sex, BMI, and TST, the timing of the sleep schedule showed significant relationships with PA (Table 2). Bedtime and wake time were positively associated with percent time spent in sedentary PA (coefficient = 3.94, $p = 0.016$ and coefficient = 3.81, $p = 0.013$, respectively). Bedtime and wake time showed significant negative associations with percent time spent in light (coefficient = –2.32, $p = 0.041$ and coefficient = –2.13, $p = 0.048$, respectively) and MVPA (coefficient = –1.62, $p = 0.013$ and coefficient = –1.68, $p = 0.005$, respectively). The midpoint of sleep showed a significant positive association with percent time spent sedentary (coefficient = 3.99, $p = 0.013$) and significant negative

**Table 2**
Multiple regression analyses showing associations between sleep measures and physical activity levels in healthy, free-living adults after adjusting for age, sex, body mass index, and TST.

| Predictor | Sedentary | | | Light | | | MVPA | | |
|---|---|---|---|---|---|---|---|---|---|
| | Coefficient | Standard error | $p$ value | Coefficient | Standard error | $p$ value | Coefficient | Standard error | $p$ value |
| Bedtime | 3.94 | 1.46 | **0.016**[a] | –2.32 | 1.05 | **0.041**[b] | –1.62 | 0.58 | **0.013** |
| Wake time | 3.81 | 1.36 | **0.013**[a,c] | –2.13 | 1.00 | **0.048**[b] | –1.68 | 0.52 | **0.005**[d] |
| Midpoint of sleep | 3.99 | 1.42 | **0.013**[a] | –2.29 | 1.03 | **0.041**[b] | –1.70 | 0.55 | **0.007** |
| MEQ score | 0.31 | 0.22 | 0.17 | 0.17 | 0.15 | 0.28 | 0.14 | 0.09 | 0.11 |
| ESS score | 0.28 | 0.83 | 0.74 | 0.05 | 0.56 | 0.94 | –0.32 | 0.32 | 0.34 |
| PSQI score | –1.03 | 1.78 | 0.57 | 0.95 | 1.20 | 0.44 | 0.08 | 0.72 | 0.91 |

*Abbreviations:* MEQ, Morningness–Eveningness Questionnaire; MVPA, moderate-to-vigorous physical activity; ESS, Epworth Sleepiness Scale; PSQI, Pittsburgh Sleep Quality Index; TST, total sleep time.
Significant results ($p < 0.05$) are indicated in bold.
[a] Indicates significant effect of age covariate (coefficients = –0.83 to –0.85, $p$ values < 0.04).
[b] Indicates significant effect of age covariate (coefficients = 0.58–0.59, $p$ values < 0.05).
[c] Indicates significant effect of TST covariate (coefficient = –0.13, $p = 0.027$).
[d] Indicates significant effect of TST covariate (coefficient = 0.05, $p = 0.032$).

associations with percent time spent in light (coefficient = –2.29, $p = 0.041$) and MVPA (coefficient = –1.70, $p = 0.007$). In other words, later bedtime, wake time, and midpoint of sleep are all associated with more time spent in sedentary PA and less time spent in light PA and MVPA. Conversely, earlier bedtime, wake time, and midpoint of sleep are associated with less time spent in sedentary PA, and more time spent in light and MVPA. A trend for a positive association between MEQ score and MVPA was found (coefficient = 0.14, $p = 0.11$), indicating higher percent time spent in MVPA in earlier chronotypes and lower percent time spent in MVPA in later chronotypes (Table 2). When participants were categorically divided into those having lower MEQ scores and those having higher MEQ scores by median split, those in the higher MEQ group (i.e., morning type) were found to have spent a significantly higher percentage of time in MVPA compared to those in the lower MEQ group (i.e., evening type; 4.64% vs. 1.99%; $p = 0.045$ by $t$-test). No differences were observed in the low versus high MEQ score subgroups in percentage of time spent in sedentary (85.07% vs. 80.86%, $p = 0.25$) or light PA (12.94% vs. 14.49%, $p = 0.54$). No significant relationships were observed between ESS, PSQI, and any PA measures (Table 2).

In considering the covariates that were used to adjust the multiple regressions, we observed that age was also related to PA levels when considered together with bedtime, wake time, and midpoint of sleep (Table 2). Specifically, age was found to have a negative association with percent time spent in sedentary PA and a positive association with percent time spent in light PA. In the models considering wake time, the waist-derived measure of TST was negatively associated with percent time spent in sedentary PA and positively associated with percent time spent in MVPA. In these cases, shorter sleep duration was associated with more time spent in sedentary PA and less time spent in MVPA.

Sex was not related to sleep timing and PA. Furthermore, when an exploratory secondary analysis was run with only males ($n = 16$), the results were similar to when both males and females were included. Specifically, significant relationships were seen for bedtime, wake time, and midpoint of sleep with percent time spent in sedentary PA and MVPA ($p$ values $< 0.05$). The observed relationships between bedtime and midpoint of sleep with percentage of time spent in light PA were reduced to a trend ($p = 0.06$ and $p = 0.051$, respectively), likely as a result of the smaller sample size.

## 4. Discussion

Here, we demonstrate that a slightly delayed individual sleep timing schedule is associated with increased time spent in sedentary PA and less time spent in MVPA in adults. Although values for this small sample size did not reach statistical significance, it appears that later chronotype may also be associated with lower MVPA. It should also be noted that while the two are correlated, chronotype may be reflective of underlying circadian physiology, whereas sleep timing is a behavior influenced by societal demands. This may also account for the lack of a significant association between chronotype and PA. Nevertheless, current findings suggest that even relatively small alterations in sleep timing may influence PA.

A study by Baron et al. demonstrated a relationship between delayed sleep and increased caloric intake and BMI, although average 24-h PA was not correlated with sleep timing or duration [16]. Current findings are consistent with a study showing that children/adolescents with late bedtime/wake time had lower PA than those with early bedtime/wake time, despite a 7-min between-group difference in sleep duration [7]. To our knowledge, the present report is the first to demonstrate a relationship between sleep timing and PA in adults.

It appears that short sleep duration is also affecting the levels of sedentary PA and MVPA, especially when considered in conjunction with delayed wake time. This observation is conceptually in line with the reported link between short sleep duration and obesity [1]. Interestingly, the lack of an effect of TST when included in the bedtime models may indicate a relatively more important role of bedtime than wake time in influencing free-living PA levels. Delaying bedtime may preferentially expose individuals to light during the later hours of the day, which was recently found to correlate with higher BMI [17].

Some limitations should be addressed. This was a retrospective cross-sectional analysis of data collected during a screening period. A controlled trial that manipulates sleep timing to determine whether PA levels are changed would better address the question. The sample size was small. However, a similar relationship between late sleep timing and low MVPA was demonstrated with a large sample ($n = 2200$) of children/adolescents [7]. Despite having results that are consistent with that of the larger study, there may remain the risk for a type II error due to the small number of participants in this study. However, the current study may be the first to report such a relationship in adults. These findings can be considered preliminary but urge more work on the topic. The small sample likely resulted in a homogeneous sample with limited chronotype variation. These results may not be directly applicable to individuals with extremely late bedtimes or delayed sleep-phase disorder. Therefore, more work should be done in a larger sample size to confirm the current findings.

In the current study, actigraphs were mounted on the waist. While this is the ideal location for determining PA, the validated site for sleep is the nondominant wrist [18]. However, in a comparison of actigraphs simultaneously recording from both sites, the waist scoring agreed with the wrist scoring in 95.6% of the sleep minutes [19]. In the same study, sleep duration estimates between the two sites were significantly correlated, with an observed mean difference of –1.07 min [19]. Nevertheless, we chose to include the waist-derived measure of TST only as a covariate in the multiple regression models, in an attempt to objectively control for the effects of sleep duration, but not as a primary predictor variable.

The present findings identify sleep timing as a lifestyle behavior that may influence energy expenditure. Here, we report novel findings that delayed sleep timing is associated with lower PA levels in adults. This is consistent with prior findings that have identified worsened behaviors for cardio-metabolic health within individuals with delayed sleep. Specifically, shift work is associated with increased smoking and sedentary behavior [20]. Preference for delayed sleep timing was also associated with increased smoking [21], and greater consumption of alcohol [21], caffeinated soft drinks [21], and sugar-sweetened soda [16]. While still speculative, due to the cross-sectional nature of the study, current findings may indicate a putative pathway by which delayed sleep timing also results in reduced daytime PA. More work should be done to assess the relationship between sleep timing and weight status, including thorough laboratory-based experimental intervention studies.

## Funding sources

This study was supported by grants from the National Institutes of Health R01HL091352 (St-Onge) and T32DK007559 (Shechter), a St. Luke's/Roosevelt Pilot and Feasibility Grant (St-Onge), and a New York Obesity Research Center Grant (P30DK26687). The study was also supported in part by Columbia University's CTSA grant No. UL1 TR000040 from NCATS/NIH. The content is solely the responsibility of the authors and does not necessarily represent the official views of the NIH.

## Conflict of interest

None of the authors have any conflict of interest to disclose.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2014.07.010.

## Acknowledgments

We thank Amy L. Roberts for her help in data collection.

## References

[1] Patel SR, Hu FB. Short sleep duration and weight gain: a systematic review. Obesity (Silver Spring) 2008;16(3):643–53.
[2] St-Onge MP, O'Keeffe M, Roberts AL, RoyChoudhury A, Laferrere B. Short sleep duration, glucose dysregulation and hormonal regulation of appetite in men and women. Sleep 2012;35(11):1503–10.
[3] St-Onge MP, Roberts AL, Chen J, Kelleman M, O'Keeffe M, RoyChoudhury A, et al. Short sleep duration increases energy intakes but does not change energy expenditure in normal-weight individuals. Am J Clin Nutr 2011;94(2):410–16.
[4] Shechter A, Rising R, Albu JB, St-Onge MP. Experimental sleep curtailment causes wake-dependent increases in 24-h energy expenditure as measured by whole-room indirect calorimetry. Am J Clin Nutr 2013;98(6):1433–9.
[5] Golley RK, Maher CA, Matricciani L, Olds TS. Sleep duration or bedtime? Exploring the association between sleep timing behaviour, diet and BMI in children and adolescents. Int J Obes (Lond) 2013;37(4):546–51.
[6] Adamo KB, Wilson S, Belanger K, Chaput JP. Later bedtime is associated with greater daily energy intake and screen time in obese adolescents independent of sleep duration. J Sleep Disorders Ther 2013;2(4):1–5.
[7] Olds TS, Maher CA, Matricciani L. Sleep duration or bedtime? Exploring the relationship between sleep habits and weight status and activity patterns. Sleep 2011;34(10):1299–307.
[8] Chaput JP. Sleep patterns, diet quality and energy balance. Physiol Behav 2013;doi:10.1016/j.physbeh.2013.09.006.
[9] Shechter A, O'Keeffe M, Roberts AL, Zammit GK, RoyChoudhury A, St-Onge MP. Alterations in sleep architecture in response to experimental sleep curtailment are associated with signs of positive energy balance. Am J Physiol Regul Integr Comp Physiol 2012;303(9):R883–9.
[10] Freedson PS, Melanson E, Sirard J. Calibration of the Computer Science and Applications, Inc. accelerometer. Med Sci Sports Exerc 1998;30(5):777–81.
[11] Tucker JM, Welk GJ, Beyler NK. Physical activity in U.S.: adults compliance with the Physical Activity Guidelines for Americans. Am J Prev Med 2011;40(4):454–61.
[12] Sadeh A, Sharkey KM, Carskadon MA. Activity-based sleep-wake identification: an empirical test of methodological issues. Sleep 1994;17(3):201–7.
[13] Horne JA, Ostberg O. A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. Int J Chronobiol 1976;4(2):97–110.
[14] Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep 1991;14(6):540–5.
[15] Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res 1989;28(2):193–213.
[16] Baron KG, Reid KJ, Kern AS, Zee PC. Role of sleep timing in caloric intake and BMI. Obesity (Silver Spring) 2011;19(7):1374–81.
[17] Reid KJ, Santostasi G, Baron KG, Wilson J, Kang J, Zee PC. Timing and intensity of light correlate with body weight in adults. PLoS ONE 2014;9(4):e92251.
[18] Hjorth MF, Chaput JP, Damsgaard CT, Dalskov S-M, Michaelsen KF, Tetens I, et al. Measure of sleep and physical activity by a single accelerometer: can a waist-worn Actigraph adequately measure sleep in children? Sleep Biol Rhythms 2012;10:328–35.
[19] Paavonen EJ, Fjallberg M, Steenari MR, Aronen ET. Actigraph placement and sleep estimation in children. Sleep 2002;25(2):235–7.
[20] Johansson G, Johnson JV, Hall EM. Smoking and sedentary behavior as related to work organization. Soc Sci Med 1991;32(7):837–46.
[21] Wittmann M, Dinich J, Merrow M, Roenneberg T. Social jetlag: misalignment of biological and social time. Chronobiol Int 2006;23(1–2):497–509.